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Implications for treating male depression

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Factor Structure of the Gotland Scale of Male Depression in Two Samples of Men With Prostate Cancer: Implications for Treating Male Depression

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Christopher F. Sharpley, PhD^{1,2}, Vicki Bitsika, PhD²,
David R. H. Christie, MB, ChB¹, and Myra S. Hunter, PhD³

Abstract

Up to a quarter of all prostate cancer (PCa) patients suffer from clinically significant depression but treatments are inconsistent and short-lived in their efficacy. One possible reason could be that “male depression” is not adequately diagnosed by the criteria for major depressive disorder (MDD) used in many clinical settings. In response to this limitation, the Gotland Scale of Male Depression (GSMD) was developed to identify the extra symptoms of MDD in men. Although the factor structure of the GSMD has been reported in non-PCa samples, it has not been determined for this group of men. Two samples of PCa patients were recruited, 191 from Australia and 138 from the United Kingdom and all patients received the GSMD individually, plus a background questionnaire. Two-factor solutions were identified for each of the two samples. The Australian sample was characterized by changes in emotional and somatic function, followed by depressed mood. The U.K. sample exhibited the same two-factor solution but in reverse order of weighting. Targeted treatments for depression in PCa patients may benefit from identification of the loadings that individual patients have on these two GSMD factors so that specific clinical profiles and treatment needs may be based on this information about their depression.

Keywords

depression, male depression, prostate cancer, diagnosis, treatment

Introduction

Although significant numbers of prostate cancer (PCa) patients become depressed (Bill-Axelsson et al., 2011; Johansson et al., 2011; Kunkel, Bakker, Myers, Oyesanmi, & Gomella, 2000) and experience higher rates of admission to emergency treatment, hospitalization, outpatient visits, and death (Jayadevappa, Malkowicz, Chhatre, Johnson, & Gallo, 2011), two recent reviews of interventions used to reduce this depression reported only partially supportive results for those interventions. Newby, Graff, Ganzani, and McDonagh (2015) applied meta-analysis to data from nine studies that used exercise, information, psychotherapy, and peer support as treatment for depression among PCa patients and not only reported an overall significant reduction in depression but also noted that only one of the nine studies reported a statistically significant improvement in patients' depression scores. In their systematic review of 14 randomized control studies of interventions to relieve anxiety and depression among

PCa patients, Chien, Liu, Chien, and Liu (2014) reported that psychosocial strategies significantly reduced depression in these patients but only for a period of 3 months after treatment.

Although the most commonly used classification for depression, the diagnostic criteria for major depressive disorder (MDD) set out in the fifth edition of the *Diagnostic and Statistical Manual for Mental Disorders (DSM-5; APA, 2013)* may not be completely valid when measuring depression in men because men may exhibit additional symptoms of depression, as demonstrated by the Gotland Study of the effects of general practitioner

¹University of New England, Armidale, New South Wales, Australia

²Bond University, Robina, Queensland, Australia

³King's College, London, UK

Corresponding Author:

Christopher F. Sharpley, Brain-Behaviour Research Group, University of New England, Armidale, NSW 2351, Australia.

Email: csharp13@une.edu.au

educational training on suicide rates (Rutz, von Knorring, Pihlgren, Rihmer, & Walinder, 1995). In the Gotland Study, the overall suicide rate decreased by 60% after general practitioners had been trained to recognize the symptoms of MDD but that change in suicide rate was due to decreases in the female suicide rate but not in the male suicide rate. Rutz et al. (1995) argued that this was due to the underdiagnosis of men who were depressed but not easily identifiable via MDD symptomatology alone and that *extra symptoms* of depression were needed when measuring depression in males (Innamorati et al., 2011; Rutz et al., 1995; Zierau, Bille, Rutz, & Bech, 2002), including aggression, irritability and alcohol use, none of which are included in the diagnostic criteria for MDD (APA, 2013).

If this “male depression” hypothesis is true, then it may at least partially account for the inconsistent and overall limited effectiveness of the interventions reviewed by Newby et al. (2015) and Chien et al. (2014) because “true” male depression in PCa patients may not have been reliably assessed by the instruments used in the studies examined in those reviews. Some supporting evidence for this argument comes from a previous study which assessed the prevalence of depression via the Gotland Scale for Male Depression (GSMD; Sharpley, Bitsika, & Christie, 2014), which was developed to identify and assess the severity of MDD plus symptoms of male depression. In that study, about 24% of the PCa patients who were identified as depressed on the GSMD would not have been similarly identified by the application of the diagnostic criteria for MDD alone (Sharpley, Bitsika, & Christie, 2014).

However, depression is not a unitary construct, with at least 1,497 possible combinations for MDD (Ostergaard, Jensen, & Bech, 2011), adding impetus to the call for individualized assessment and treatment of depression (Insel, 2013), and it may be that male depression as measured by the GSMD is similarly multidimensional in its structure. Several previous studies of the GSMD have explored its underlying factor structure in an attempt to further describe the underlying structure of the male depression construct. Innamorati et al. (2011) examined the GSMD factor structure in 152 male psychiatric inpatients and reported a single-factor solution, as did Chu et al. (2014) with 231 male outpatients from a polyclinic, but no previous reports have been made of the factor structure of the GSMD in PCa patients. Because this information may help identify aspects of the GSMD that can be targeted for specific treatment options, thus potentially increasing the efficacy of therapy for depression in PCa patients, this study explored the factor structure of the GSMD in PCa patients. To enhance the generalizability of the findings, two samples were recruited, each from a different nation and data from each were examined

separately and then compared for their overall results in order to emphasize any national differences that may occur.

Method

Subjects

A total of 329 men with PCa were recruited, 191 (58.0%) from the Gold Coast, Australia and 138 (42.0%) from London, United Kingdom. All were volunteers and were attending outpatient clinics. They were recruited by administration staff, cancer nurses, radiologists, or oncologists either while waiting for treatment or via a telephone and postal survey.

Materials

Demographic Questionnaire. A background questionnaire about participants' ages, living situation, time since diagnosis, present status of their PCa and past and present treatments. Although there are several possible hormone treatments, some of which may vary in their effects, this variable was aggregated here due to the small number of patients who were receiving some specific hormone treatments.

Male Depression. GSMD was developed by Rutz and colleagues (1995) to improve recognition of depression in males. The GSMD has satisfactory validity against the Major Depression Inventory and internal consistency (Cronbach's alpha) of .86 (Zierau et al., 2002). Respondents to the GSMD are asked to indicate “if your behaviour has changed during the last two weeks” in any of 13 directions, each of which is measured by a single GSMD item, and to rate the intensity of their responses as “Not at all,” “To some extent,” “Very true,” and “Extremely so.” The GSMD is composed of two subscales, one which assesses distress (seven items) and one which assesses depression (six items). Although five of the six depression items match DSM-5 criteria for MDD, the final item asks about the respondent's family's “tendency towards abuse, depression/dejection, suicide attempts” rather than about the respondent himself. Scores on the GSMD range from 0 to 39 and may be broken down into: 0 to 12 = *no depression*, 13 to 26 = *probable depression*, and 27 to 39 = *definite depression requiring treatment* (Zierau et al., 2002).

Statistical Analysis

Data were analyzed via SPSS 22.0. Descriptive data were calculated and reliability of the GSMD assessed. Correlation coefficients were calculated to investigate the

Table 1. Demographic and Gotland Scale of Male Depression (GSMD) Data for Two Samples.

Variable	Australian sample	U.K. sample	F	p
Mean age, years	69.59	70.22	0.664	.416
Living situation (%)				
With wife/partner	87.8	82.6		
Widowed	2.1	4.3		
Divorced/separated	4.8	8.0		
Never married	5.3	5.1		
Mean time since diagnosis, months	26.99	19.32	5.819	.016
Present cancer status (%)				
Still present	25.3	60.1		
Remission	68.8	28.2		
Recurring	5.9	11.6		
Past treatment (%)				
Radiotherapy	16.6	27.5		
Surgery	7.5	8.7		
Hormone	3.7	9.4		
Combinations	58.9	54.4		
None	13.4	0.0		
Current treatment (%)				
Radiotherapy	2.7	14.5		
Surgery	0.0	0.0		
Hormone	21.4	25.4		
Combinations	3.7	60.1		
None	72.2	0.0		
GSMD, mean (SD)	4.575 (5.490)	4.93 (5.03)	0.304	.582
GSMD categories (%)				
No depression	89.0	92.0		
Probable depression	11.0	8.0		
Requiring treatment	0.0	0.0		

associations between the demographic variables and GSMD scores; multivariate analysis of variance was used to test for the presence of significant differences in age, time since diagnosis, and GSMD score across the two samples. Exploratory factor analysis was conducted to determine the underlying structure of the GSMD across each sample. Principal components analysis was used in the identification of item clusters and factor loadings generated using both orthogonal and oblique rotations to determine the most coherent and interpretable solution.

Results

Characteristics of Study Participants

The demographic and GSMD characteristics of the two samples are reported in Table 1. Internal consistency (Cronbach's alpha) for the GSMD was satisfactory for the Australian (.875) and U.K. (.851) samples and the deletion of no GSMD item would have increased these values by more than 2.0%, therefore all GSMD items were included at this stage of the analysis. The only significant correlation between any of the demographic variables

and the GSMD total score was for age (Australian sample, $r = -.227$, $p < .01$; U.K. sample, $r = -.225$, $p < .01$), indicating that male depression significantly decreased with age across both samples, as in some other data (Henderson et al., 1998). The Australian patients had received their PCa diagnoses a significantly longer time than the U.K. sample (Table 1) but this was not significantly associated with the GSMD total scores. The relative proportions of samples that scored in the three depression categories for the GSMD are reported in Table 1.

Factor Analysis

Australian Sample. There were many interitem correlations greater than .3 between the GSMD Items 1 to 12 but none for GSMD Item 13, which was therefore deleted from the rest of this factor analysis (as Innamorati et al., 2011, also did for the same reason). Kaiser and Rice's (Kaiser, 1974) Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was 0.878 and Bartlett's test of sphericity was significant ($\chi^2 = 1241.31$, $p < .001$), thus justifying factor analysis with these data (Tabachnik & Fidell, 2007). Principal components extraction revealed two factors with

eigenvalues greater than 1.0, verified by inspection of the scree plot and parallel analysis. These two factors accounted for 59.19% of the variance (Factor 1 had an eigenvalue of 6.025 and accounted for 50.206% of the variance, Factor 2 had an eigenvalue of 1.079 and accounted for 8.988% of the variance). Oblimin rotation confirmed this two-factor solution.

U.K. Sample. Again, GSMD Item 13 did not correlate strongly with the remaining 13 items and was deleted from further analysis. There were multiple interitem correlations in excess of .3 among the remaining 12 GSMD items, the KMO was 0.855, and Bartlett's test of sphericity was significant ($\chi^2 = 704.501$, $p < .001$). There were two factors with eigenvalues greater than 1.0, verified by the scree plot and parallel analysis. These two factors accounted for 55.175% of the variance (Factor 1 eigenvalue = 4.967, 41.388% of the variance; Factor 2 eigenvalue = 1.655, 13.789% of the variance). Oblimin rotation also confirmed this two-factor solution.

The item loadings from the pattern matrices for the Australian and U.K. sample solutions are reported in Table 2 and indicate that a two-factor solution, rather than a single-factor solution as reported by Innamorati et al. (2011) and Chu et al. (2014) most appropriately fits these data consistently across the two samples of PCa patients, although the order of the factors is reversed from the Australian to the U.K. sample. The first factor in the Australian sample (and the second factor in the U.K. sample) may be defined as "Change in Emotional and Somatic Functioning" and the second factor in the Australian sample (first factor in the U.K. sample) may be defined as "Depressed Mood." The interfactor correlation was .691 for the Australian sample and .596 for the U.K. sample, both $p < .001$ and accounting for 35.5% and 47.7% of the variance, respectively, arguing that these two factors are connected but remain discrete. Neither of these two factors was significantly correlated with time since diagnosis, present cancer status, and past or current treatment in the Australian sample but they were both significantly correlated with age (Factor 1, $r = -.226$, $p = .002$; Factor 2, $r = -.183$, $p = .013$). For the U.K. sample, there were no significant correlations between Factors 1 or 2 and time since diagnosis, present cancer status, and past or current treatment but Factor 2 was significantly correlated with age in this sample ($r = -.246$, $p = .004$).

As reported in Table 2, the content of the two factors is not completely congruent across the two samples, with Factor 1 including Decision making and Aggression in the Australian sample but not in the U.K. sample, and Factor 2 including Burned out and Fatigue for the U.K. sample but not for the Australian sample. These minor differences reflect those that are common across factor structures based on different samples (Tabachnick &

Table 2. Factor Structure and Gotland Scale of Male Depression (GSMD) Items Loadings for Australian and U.K. Patients.

GSMD item content	Factor 1	Factor 2
Australian sample		
1. Fatigue	.853	
2. Stress	.822	
3. Burned out	.804	
4. Irritability	.779	
5. Anxious	.706	
6. Sleep problems	.705	
7. Decision making	.679	
8. Aggression	.585	
9. Alcohol, drugs, hyperactivity		.732
10. Gloomy		.671
11. Behavior changed		.595
12. Self-pity		.479
U.K. sample		
1. Gloomy	.905	
2. Behavior changed	.873	
3. Self-pity	.772	
4. Aggression	.690	
5. Decision making	.553	
6. Alcohol, drugs, hyperactivity	.540	
7. Burned out		.910
8. Fatigue		.887
9. Irritability		.741
10. Anxious		.576
11. Sleep Problems		.552
12. Stress		.548

Fidell, 2007) but, overall, the two factor structures are quite similar across the two samples.

Discussion

The presence of a two-factor solution in both these samples of PCa patients compared with previous reports of a single-factor solution in non-PCa men argues for consideration of depression among PCa patients as being unique and requiring targeted treatment models that may be different to those applied to non-PCa populations. In addition, this variability in factor structure between those previously reported in non-PCa samples of men (i.e., psychiatric patients: Chu et al., 2014, Innamorati et al., 2011) and these two samples of PCa patients may be a possible reason for the relatively poor efficacy of standardized treatments for depression in this patient group that was reported by the two reviews of PCa depression mentioned in the "Introduction" section of this article (Chien et al., 2014; Newby et al., 2015). This kind of variability in factor structures drawn from different samples has been previously noted (Tabachnick & Fidell, 2007) and needs to

be considered when applying results of studies on a particular population to diagnosis and treatment planning within a different population, particularly across patients with an identifiable physical disease (such as PCa) and purely psychiatric patients (on which most evidence supporting standardized treatments for depression is based).

Although the same two factors were identified in each sample, the variability in loading of those factors suggests that U.K. PCa patients may differ from their Australian counterparts in the ways that depression manifests itself. Depression in the Australian men was strongly characterized by changes in their physiological and emotional states but lesser in their symptoms of depressed mood, whereas their U.K. peers exhibited depression more strongly characterized by feeling sad and depressed with less influence from somatic and emotional changes. These differences in factor loadings also suggest that similar differences might be present across other national or ethnic populations, and further emphasize the need to base treatment planning on ideographic data rather than assuming that all men experience depression in identical ways, regardless of their social and cultural backgrounds or disease status.

Limitations of this study include the social and cultural nature of the two samples (which may not only be similar in many ways but also hold specific differences that may have contributed to the different results from the United Kingdom and Australia), the use of the GSMD only once, and the relatively shallow nature of the background questionnaire which did not include social and economic data which could influence depressive status. Although the initial analyses conducted on these data did not reveal any significant association between male depression and cancer status, or past and current treatment, the latter variables were relatively confined and thus cannot be ruled out of contention as possible causal factors in the development and severity of male depression. Although self-reports of depressive symptoms have a sound relationship with structured clinical interviews, application of the latter could provide valuable information regarding the details of depression among these men. Because of the small numbers of patients receiving some forms of hormone treatments, the presence of different effects from different hormone treatments was not examined but is a potentially fruitful avenue for further research because of the previous research which demonstrated depressive effects from such treatments (Sharpley, Christie, & Bitsika, 2014).

In summary, assessment of depression in PCa patients may benefit from an awareness of the underlying structure of that depression and how it may manifest as more than simply MDD symptoms. Using a standardized MDD-based treatment model with all PCa patients is less likely to be effective than individualized treatment plans based on the specific symptom profiles of these patients, and the

application of the GSMD could provide valuable data for provision of personalized treatment for the depression that PCa patients experience (Insel, 2013), thus potentially increasing the (currently low) efficacy of antidepressant interventions with these men. Furthermore, examination of the underlying structures of male depression as it is experienced by particular disease- and national/cultural groups of men may assist in developing more targeted and effective treatments for male depression.

Declaration of Conflicting Interests

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